

WHAT'S NEW IN INTENSIVE CARE



How we use ultrasound to support clinical decisions on fluid administration in critical ill patients

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Intravenous fluid administration is a common critical care intervention and fluids should be treated as drugs with specific indications [1]. Traditionally, fluid therapy focussed mainly on increasing cardiac output. However, fluid administration can have harmful consequences, such as pulmonary or visceral interstitial oedema, which worsens organ dysfunction. In this context, critical care ultrasound (CCUS) helps to assess potential risks and benefits of fluid administration. We describe our use of CCUS to support clinical decisions regarding fluid administration, focussing primarily on the optimization and stabilisation phase of the critically ill patient.

A framework for clinical decision-making on fluids

Fluids should be considered as therapeutic option when signs of tissue hypoperfusion are present. In this context, CCUS offers three key tools—critical care echocardiography (CCE), lung ultrasound (LUS), and venous excess ultrasound (VExUS)—which, when combined, can help answer important questions about risks and benefits of fluid administration. A holistic step-by-step approach for clinical decision-making is provided (Fig. 1):

1. *Are there signs of overt hypovolemia?* CCE allows a rapid evaluation of whether there is overt hypovolemia by (even visually) assessing the end-diastolic volumes and the systolic function of the left

ventricle (LV). In absence of a dilated right ventricle (RV), an LV end-diastolic area (LVEDA) $< 10 \text{ cm}^2$ in parasternal (or subcostal) short axis at the level of papillary muscles together with an end-systolic cavity obliteration (“kissing walls”) is a strong indicator of overt hypovolaemia [2]. LVEDA may help differentiate hypovolemia (decreased LVEDA) from vasoplegia (normal LVEDA). The presence of overt hypovolemia may be confirmed by a small end-expiratory inferior vena cava (IVC_{ee}) $\leq 10 \text{ mm}$ [3]. Advanced CCE may reveal LV outflow tract (LVOT) obstruction in some patients with overt hypovolemia [4]. In case of overt hypovolemia and impending cardiovascular collapse, fluids should be given immediately to restore perfusion, without delay for further risk assessment or fluid-responsiveness testing.

2. *Are there signs of critical cardiac conditions?* In patients with absence of overt hypovolaemia, CCE allows to screen for cardiac conditions associated with an increased risk for pulmonary or systemic congestion, such as a dilated RV (RV/LV end-diastolic ratio > 0.6), presence of severe valve disease, or presence of increased LV filling pressures (LVFP). In basic CCE, a severely depressed LVEF is usually indicative of increased LVFP. Left atrial enlargement and a fixed right-bowing of the interatrial septum strongly suggests chronically elevated LVFP, if conditions such as atrial arrhythmias and mitral valve disease are excluded. Using advanced CCE, conventional and tissue Doppler at mitral valve level provide semi-quantitative measures of LVFP. “The rule of 8’s”, which means a lateral ratio of early transmitral flow velocity to early diastolic myocardial veloc-

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ity (E/e') > 8 and/or a lateral $e' \leq 8$ cm/s, may help to identify patients with possibly raised LVFP [5]. A ratio of early to late diastolic transmitral flow velocity (E/A) > 1.8 usually confirms elevated LVFP with high specificity [6].

3. *Are there signs of pulmonary congestion?* LUS allows to further assess the risk of pulmonary congestion [7]. A bilateral A-profile (< 3 B-lines per view), indicates a low LVFP with high certainty. In this case, if fluids are indicated, the risk of pulmonary congestion is low. Conversely, in case of a bilateral B-profile (≥ 3 B-lines), a wide range of LVFP can be expected [8]. A homogenous distribution of B-profiles with a smooth pleural line is suggestive of hydrostatic pulmonary oedema, whereas a more heterogenous distribution with pleural abnormalities is more suggestive of permeability oedema [9]. Therefore, a bilateral B-profile with a heterogeneous distribution and abnormal pleural line, whilst precluding conclusions about the need for fluids, suggests the risk of precipitating further oedema after fluid administration. Additionally, medium–large bilateral pleural effusions should be considered before fluid administration, as it has been associated with higher LVFP in patients with reduced LVEF [10].
4. *Are there signs of systemic congestion?* VExUS is an emerging tool to identify and quantify systemic venous congestion, a surrogate of increased organ afterload that in turn can impair organ function due to decreased tissue-perfusion [11]. In case of a dilated $IVC_{ee} \geq 20$ mm, at least two of the following patterns are suggestive of a high risk of systemic congestion: flow reversal during systole in the hepatic vein, flow variability $> 50\%$ during the cardiac cycle in the portal vein, or discontinuation of flow with only a diastolic phase seen in renal vein. As VExUS captures the dynamic interplay between upstream venous pressure and right atrial pressure, it can support a more personalised fluid management. Whilst there is no

conclusive evidence yet, integrating VExUS into the clinical decision-making may offer additional insights without evident harm when used judiciously.

5. *What is the expected efficacy of a fluid bolus?* After excluding overt hypovolemia and subsequently assessing the presence of conditions that may warrant caution in fluid administration (LV or RV pathology, and pulmonary and/or systemic congestion), clinicians should consider the potential efficacy of a fluid bolus by evaluating fluid responsiveness, rather than adhering rigidly to the published thresholds for each parameter. The change in the velocity–time integral (VTI) of the LVOT directly reflects changes in stroke volume and can be utilised in various functional tests to assess the anticipated efficacy of a fluid bolus. In this regard, the magnitude of LVOT-VTI increase during a passive leg raise (PLR) test may directly reflect the expected efficacy of the fluid bolus. As an alternative, the sum of the absolute changes of LVOT-VTI during a combined consecutive end-inspiratory occlusion and end-expiratory occlusion test [12, 13], or the respiratory variations of the superior vena cava, are valid alternatives to predict the efficacy of a fluid bolus [14].

Clinical integration

CCUS can help critical care physicians to better balance the risks and benefits of fluid therapy by addressing key clinical questions in a structured manner. In patients without severe systolic dysfunction, signs of elevated LVFP, or congestion, fluids are generally safe if indicated. However, in the presence of these conditions, the decision to administer fluids should take into account both the potential harm and the likely benefit of a fluid bolus, considering not just the classic concept of fluid responsiveness according to fixed thresholds. Indeed, fluid decisions must consider the full clinical context of the patient.

(See figure on next page.)

Fig. 1 A holistic step-by-step approach to the use of critical care ultrasound (CCUS) to support clinical decisions regarding fluid administration is provided. Intravenous fluids should only be considered as a therapeutic option when signs of tissue hypoperfusion are present. CCUS is used to answer clinical questions (speech bubbles). If overt hypovolemia is not present, clinical questions on risk assessment (red speech bubbles) and potential benefit of fluids (green speech bubble) are answered. Thereafter, potential risks need to be weighed against potential benefits to support clinical decision-making regarding fluids in critically ill patients at the bedside. *Notably, at least three LVOT-VTI measurements should be averaged before and after functional tests such a PLR to ensure sufficient precision [15]. In the absence of an adequate apical window, a VTI gathered from the RV outflow tract (RVOT-VTI) in modified parasternal short-axis view (or eventually in modified subcostal view) may be an alternative to assess the expected efficacy of the fluid bolus in terms of SV increase. *IVC*, inferior vena cava; *LV*, left ventricle; *LVOT*, left ventricular outflow tract; *RV*, right ventricle; *VTI*, velocity–time integral. Created in BioRender. Hunsicker, O. (2025) <https://BioRender.com/fefe5x7>

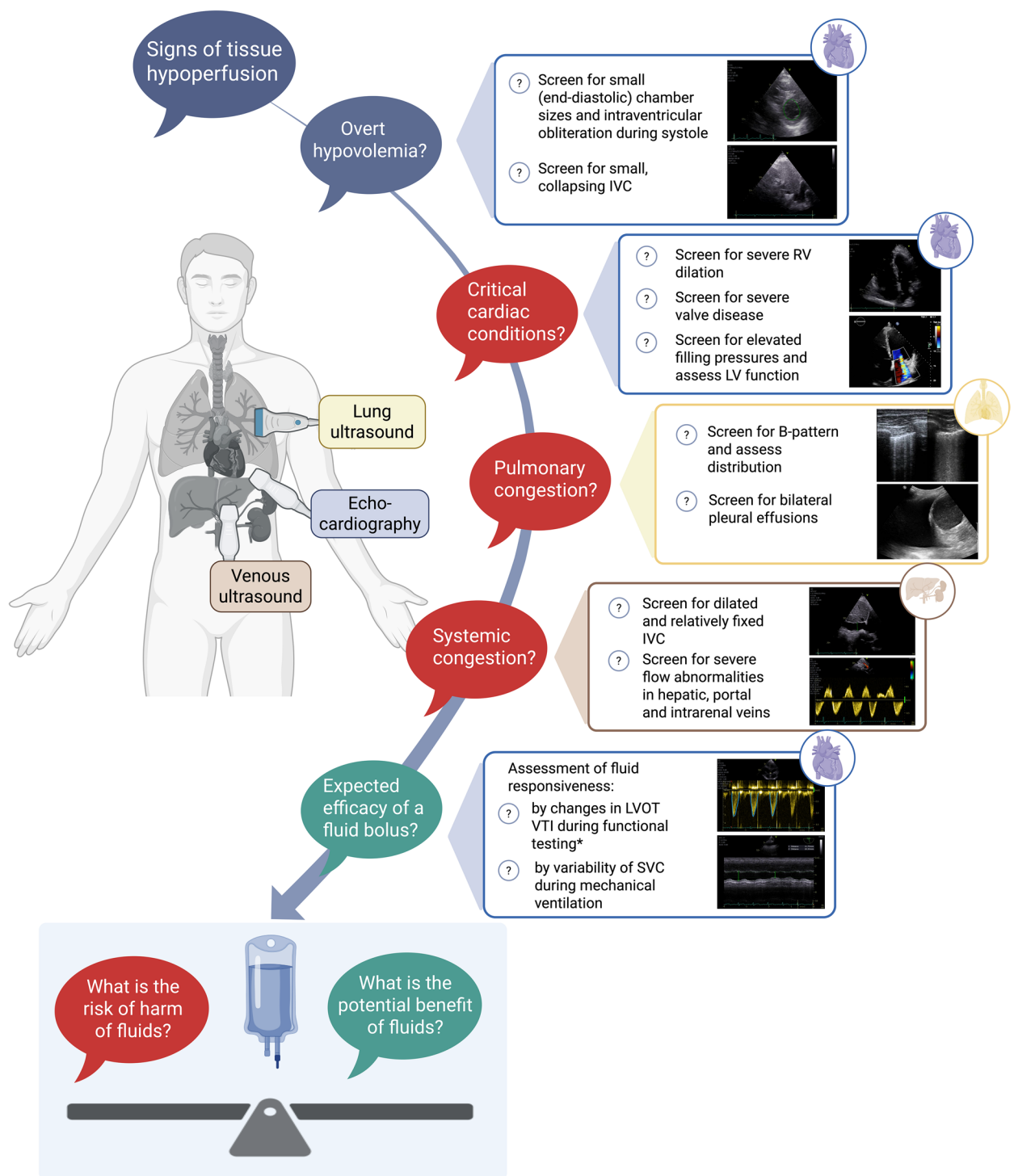


Fig. 1 (See legend on previous page.)

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Declarations

Conflicts of interest

The authors have no conflict of interest.

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